

## Unimpaired abduction to alien abduction

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## **Unimpaired Abduction to Alien Abduction:**

### **Lessons on Delusion Formation**

An examination of alien abduction belief can inform how we ought to approach constructing explanations of monothematic delusion formation. I argue that the formation and maintenance of alien abduction beliefs can be explained by a one-factor account, and that this explanatory power generalizes to (other) cases of monothematic delusions. There are no differences between alien abduction beliefs and monothematic delusions which indicate the need for additional explanatory factors in cases of the latter. I make the additional point that whilst alien abduction beliefs can be readily explained using a one-factor framework, the two-factor framework requires adjustment to accommodate them. I conclude that theorists interested in delusion formation have much to learn from the case of alien abduction belief.

#### **0. Preliminaries**

Many people believe that they have been abducted by aliens, and have, for example, been taken aboard spaceships against their will, and once there, been subjected to medical experimentation including the removal of eggs or sperm, formed sexual relationships and produced hybrid offspring with aliens, and received important information about the fate of the Earth. Estimates of the prevalence of these beliefs vary, from 'at least several thousand worldwide' (French et al. 2008: 1387) to 3.7 million in America alone (Hopkins et al. 1992, cf. French 2001 and French et al. 2008). Even the most conservative estimates should surprise us, on the assumption that these beliefs are false.<sup>1</sup>

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<sup>1</sup> I will assume that such beliefs are false. If there are in fact abductees proper the beliefs of non-proper abductees can inform the delusion formation debate, it need only be the case that some subjects who believe they have been abducted have not been. For those readers who take this note to display unwarranted caution I point out that some members of the scientific community necessitate it (e.g. Mack 1994; McLeod, Corbisier, and Mack 1996).

I argue that an investigation into these beliefs illuminates the debate on monothematic delusion formation, that is, those delusions which are ‘highly specific’ and ‘can present in isolation in people whose beliefs are otherwise entirely unremarkable’ (Coltheart, Langdon and McKay 2007: 642). I argue that we can explain the formation of abduction beliefs with a one-factor account; that is, one which, in contrast to two-factor accounts, does not have as part of its explanatory resources the claim that there is something *abnormal* occurring with the subject’s mechanisms of belief formation or evaluation.<sup>2</sup> If I am right, a more general question naturally arises: do we need to appeal to a second factor to explain other cases of bizarre belief, specifically, monothematic delusions? I argue that we do not. A methodology on which we see the target explanandum as on a continuum with more common phenomena earns its keep in this context too—one in which we are inquiring about the nature of the causal contributions to delusion formation.

The following objection may immediately occur: subjects with monothematic delusions often have damage to their lateral pre-frontal cortex, the area of the brain hypothesized to be involved in belief evaluation. This neurological damage straightforwardly speaks to the presence of a cognitive abnormality, and falsifies one-factor theories. I comprehensively address this later (§7). Suffice it to say for now that we ought not presume that the presence of this neurological abnormality settles the matter in favour of the two-factor account, lest we make the unjustified slide from *neurological* abnormality to *psychological* abnormality. The research programme of the two-factor framework is that of identifying an abnormal cognitive feature implicated in the formation or maintenance of monothematic delusions. Neurological abnormality does not yet establish the cognitive abnormality such theorists seek.

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<sup>2</sup> This kind of approach arises elsewhere in the philosophy of psychology. In the context of the debate over the nature of schizophrenic alien thought, Hanna Pickard has argued that a failure of rational control over one’s mind can lead to disownership, and she explicitly places this on a continuum alongside more common psychological phenomena (Pickard 2010).

## 1. Delusion formation

Delusions are typically characterized as formed on evidence which does not properly support their content, and maintained in the face of counterevidence or counterargument. However, prior to careful consideration of normal belief formation and maintenance processes, though common, characterizing delusions only in terms of their poor epistemic features risks begging the question in favour of two-factor accounts. If we characterize delusions in these terms, it might be taken that their poor epistemic features are ones which delineate delusional beliefs from non-delusional beliefs.<sup>3</sup> Given this, I also illuminate the phenomenon with some examples: Capgras delusion<sup>4</sup> (one's loved one has been replaced by an imposter), Cotard delusion (one is dead), and delusions of mirrored self-misidentification (the person in the mirror is not oneself).

In line with empiricist accounts of delusion formation, I assume doxasticism about monothematic delusions.<sup>5</sup> According to empiricist accounts, monothematic delusions are grounded in an abnormal experience (in contrast to rationalist accounts which have it that anomalous experiences are downstream of delusional belief and play no role in generating it, see, e.g. Campbell 2001). Within empiricism, there is disagreement with respect to the explanatory reach of such experiences. One-factor theorists claim that the only abnormality we need appeal to in explaining monothematic

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<sup>3</sup> A nearby idea is that characterizing delusions in a way which appeals to their irrationality is not to problematically assume two-factor theories because irrationality is a good indicator of cognitive abnormality. However, if the irrationality exhibited in delusion formation and maintenance is also observed in non-delusional belief formation and maintenance (see Bortolotti 2010), irrationality is not sufficient for delusional beliefs to be categorized as abnormal along this dimension (Sakakibara 2016: 148). For further discussion of the relationship between irrationality and abnormality see Sakakibara (2016) and Miyazono (2019: 55).

<sup>4</sup> Capgras delusion can occur monothematically, but it can also be part of a polythematic delusional syndrome, such as during an episode of schizophrenic psychosis (Coltheart, Langdon and McKay 2007: 642).

<sup>5</sup> It is far from settled whether monothematic delusions are beliefs (see for example Currie 2000; Egan 2009; Dub 2016 for non-doxastic positions). Although I take it that the case for doxasticism has been persuasively made (see for example Bayne and Pacherie 2005; Bortolotti 2010, 2012), it is appropriate to assume it in this context since empiricist accounts of delusion formation also start from the assumption that delusions are beliefs.

delusion formation is the anomalous experience. Two-factor theorists argue that in addition to the anomalous experience the subject has an abnormality in her mechanisms of belief formation or evaluation. The precise characterization of the second factor is what distinguishes versions of the two-factor account.<sup>6</sup>

The one-factor approach characterizes delusions as ‘rationalizations of anomalous experiences via reasoning strategies that are not, in themselves abnormal’ (Gerrans 2002: 47).<sup>7</sup> Brendan Maher defended a version of the one-factor theory claiming that ‘delusional beliefs are developed in much the same way that normal beliefs are’ (1988: 22). On Maher’s view, to explain why a subject forms a delusion, we need not appeal to any distinctive cognitive feature of the subject’s belief formation or evaluation.

In contrast, two-factor theorists point to the existence of cases of anomalous experience in the absence of delusion to motivate the search for a second clinical abnormality. This is claimed to account for the difference between subjects who have the experience and become delusional, and those who have the experience and do not. Philippa Garety and Daniel Freeman identify the second factor as a data-gathering bias (Garety and Freeman 1999, see also Huq et al., 1998; Garety et al., 1991; cf. Dudley et al., 2015; Ross et al.). Tony Stone and Andrew W. Young identify the second factor as the tendency to privilege observational data over minimizing adjustments to one’s beliefs (1997: 349–50). Martin Davies and colleagues claim that we need to explain why a delusion is *maintained*, as well as why it is *formed* initially, given its ‘implausibility and its inconsistency with everything else that the patient knows’ (Davies et al. 2001: 151). They understand the second factor in terms of a failure to inhibit a *pre-potent doxastic response*:

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<sup>6</sup> It might be thought that the view of cognition in the background of this discussion is a rather static one, talk of ‘normal cognition’ does not accommodate context (that is, someone with normal cognition in one context may have abnormal cognition in another). There are a couple of things to say here. First, in the case of monothematic delusions, it is often explicitly recognized that the beliefs are rather circumscribed and do not interact with other beliefs or behaviour (Coltheart, Langdon, and McKay 2007: 642), which suggests the recognition of context-sensitivity. Second, my argument is that alien abduction belief teaches us something about how to proceed in the delusion formation debate. Here is not the place to subject the framing of the debate to critical analysis regarding the view of cognition it adopts.

<sup>7</sup> Philip Gerrans has since abandoned his one-factor position (see Gerrans 2014).

although we usually believe what we perceive, normal subjects can ‘suspend their unreflective acceptance of veridicality and make a more detached and critical assessment of the credentials of their perceptual experiences’ in a way that subjects with delusions cannot (Davies et al. 2001: 153). In developing this view, Max Coltheart claims that damage to the right lateral prefrontal cortex introduces an abnormality in belief evaluation, and this is constitutive of a second factor (Coltheart 2005a: 154).<sup>8</sup> The implications of the presence of such neurological damage will be discussed at length later (§7).

A note on *abnormality*. I couch the discussion here in terms of *statistical* as opposed to *functional* (ab)normality. The notion of normality in play is important. Suppose that reasoning style *R* does not occur in the non-delusional population, but occurs in all and only folk with delusions. Suppose further that reasoning style *R* is functionally normal (perhaps within the range of reasoning styles within which evolution does not distinguish). A debate proceeding on statistical abnormality would take *R* to be a second factor, one proceeding on functional abnormality would not. Since functional abnormality is harder for the two-factor theorist to show (since it would require an independent account of functional normalcy), I will argue against a two-factor view employing statistical abnormality, and show that even this abnormality claim is not made good on.<sup>9</sup>

There are conceptual implications of the fact that some theorists characterize the second factor as a *deficit*, and others as a *bias*. Deficit-talk is contrary to the one-factor

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<sup>8</sup> For discussion of how various second factors help explain why a delusion is adopted rather than an epistemically better belief see (Sullivan-Bissett 2018).

<sup>9</sup> The literature is less than clear on which notion of abnormality is in play. In arguing against Maher's earlier work which had it that delusional belief formation is *rational* (Maher 1974), two-factor theorists might be read as relying on functional normality, insofar as they take the one-factor theorist's claim to be that anomalous experience is *sufficient* for delusion. In the presence of subjects with the relevant anomalous experience but without the delusional belief, the claim was that the functional abnormality which tipped the subject from experience into delusion was absent. However, recent work seeking to identify a second factor proceeds by comparing reasoning styles of delusional and non-delusional subjects and looking for differences. The work would not be complete upon finding such a difference if functional abnormality were being sought since statistical abnormality need not equal functional abnormality (fertilization of an egg by sperm illustrates this point, it is statistically abnormal but functionally normal (Millikan 1984: 34)).

theorist's claim that there is just one abnormality in play,<sup>10</sup> but it might be thought that bias-talk is consistent with the one-factor theory. There are two ways of understanding talk of reasoning *biases* in this context. The first is to understand these reasoning biases as ones that occur inside of the normal range. However, though it may well be independently interesting to get clear on the kinds of influences which are involved in delusion formation, that there are other influences is not where debate lies. If the two-factor theorist is to be understood in this way, she should relinquish that title, since the research programme of two-factor theories has centered on *abnormal* or *distinctive* cognitive factors. The second way is to understand reasoning biases as within the normal range, but as *exaggerated* or *worse* in subjects with delusions. This is incompatible with a one-factor theory. We need to keep separate the claims that there is some cognitive contribution, from the claim that there is some *abnormal* cognitive contribution. Failing to do this would make two-factor theories true simply in virtue of the fact that there is belief formation in delusion, which of course involves the presence of something cognitive. The two-factor theory is presumably meant to be more substantial than this.

One way to challenge two-factor theories would be to review the empirical literature appealed to in service of them, and argue that the cognitive processes identified by two-factor theories are not constitutive of a second factor after all (Noordhof and Sullivan-Bissett *manuscript*). My approach here is different: I will argue that we need not look for a second factor in the first place, since the one-factor account has the resources to accommodate the cognitive contributions to delusion formation and maintenance. There has then been a mis-describing of the level of explanation at which these cognitive contributions operate (that is, these contributions being *abnormal* versus their being merely *contributory*). The literature on abduction belief has *not* sought to identify a cognitive abnormality, *even though* researchers in this area recognize that there are subjects who have experiences associated with abduction beliefs, who do not form

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<sup>10</sup> Providing the identified deficit is not present in the normal population, as is the claim.

those beliefs. (Recall that it is this parallel empirical fact that motivates two-factor theories of delusion formation.) Rather, these researchers have sought to identify a variety of normal range cognitive processes that may contribute to the explanation of the generation and retention of abduction beliefs. This is a research methodology which we have no reason to doubt the explanatory value of, were we to equip ourselves with it, and turn to monothematic delusions.

## 2. Development and defence of a one-factor account

The one-factor theory has been characterized as the view that experience is *sufficient* for delusion formation, and so as falsely predicting that all those who, for example, experience a lack of affective response to familiar faces will develop the Capgras delusion (Davies et al. 2001: 144). Opponents claim that an account of delusion formation is required to explain why the same type of anomalous experience gives rise to a delusion in one subject, but not in another. A second factor causally implicated in the former subject and absent in the latter is thought to do this work.

However, one-factor theorists need not claim that anomalous experience is *sufficient* for delusion formation. Instead they can appeal to *normal range* reasoning.<sup>11</sup> Talk of *normal range* in this context latches onto the idea that the irrationality of subjects with delusions is not qualitatively different from that exhibited in ordinary belief formation. This is not to use the notion of normal range to *explain* delusion, it is not hypothesized to be a feature of delusion formation any more than a subject having oxygen in her environment is. Normal range responses to experience, like oxygen in the subject's

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<sup>11</sup> Two alternative ways to respond to this worry should be noted. First, one could argue that delusional hypotheses are in fact rational 'given the intensity of the experiences they are developed to explain' (Maher 1974: 105). Second, one could claim that putative cases of same experiences resulting in different beliefs, are not cases of the same experience after all (Maher 1999: 566; 2006: 182). (See Wilkinson 2015 for the empirical case for this claim as it applies to Capgras subjects). I discuss these lines of response more fully elsewhere (Noordhof and Sullivan-Bissett *manuscript*).



environment, are merely *in the full causal story*. The point is that reasoning *outside* of the normal range *is not* something we need in our explanatory toolbox.

Normal range individual differences can explain differences in belief based on the same kind of experience. Here I mean to capture the kinds of differences which might contribute to some subjects thinking in conspiratorial terms, opting for supernatural over naturalistic explanations, and so on. We can get a full explanation of why a delusion is formed and/or maintained by the combination of anomalous experience plus individual differences in reasoning styles which fall into the normal range for human psychology.<sup>12</sup> We thus need not posit an extra factor at this level, since falling at some place in the normal range with respect to reasoning does not require us to posit an abnormality. Rather, we have normal range reasoning applied to abnormal experiences.

Peter Clutton, Stephen Gadsby, and Colin Klein (2017) in their work on taxonomising delusions by etiology over content have spoken to the question of what counts as a *contribution* to delusion formation. They take it that something is *aetiologically relevant* to the formation of a delusion if and only if that thing can be changed which would thereby change whether the patient has the delusion (keeping everything else fixed) (2007: 519). They note that this is a very permissive account since we are usually interested in *actual* difference makers; things which *in fact* vary within a population, which *in fact* contribute to delusion formation. What is relevant to my discussion is that the one-factor approach can perfectly well accept even this permissive account of the *causal contributions* in delusion formation. Appeal to actual or potential difference makers is a perfectly legitimate way to proceed for those interested in the *full causal story* of the

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<sup>12</sup> Earlier I noted that one-factor accounts are incompatible with biases that are within the normal range but *exaggerated* or *worse*. Now I say that the one-factor account can accommodate certain reasoning styles (including biases) by appeal to the normal range. These might sound like incompatible claims. To be clear, then, I am granting to the two-factor theorist that they need not demonstrate a different *kind* of reasoning bias to substantiate their second factor, it is enough to demonstrate that there is a kind of reasoning which, although present in non-delusional subjects, is systematically *exaggerated* in subjects with delusions, such that there is a difference in degree. When I claim that one-factor theorists can capture reasoning styles as within the normal range, that is only providing that such styles are not exaggerated in *all and only the deluded*. To do the explanatory work of an account of delusion formation, I say, we need not suppose that they are.

genesis of a delusion, and in that story, the presence of oxygen will also come out as causally contributory. That is fine by me. I only claim that we need not characterize these actual or potential difference makers as *second factors*, that is, as having the property of being *clinically significant*, and hence explanatorily relevant to *delusional* belief formation in particular, unless they fall outside of the normal range.

Determining the boundaries on the normal range is a theoretical duty the one-factor theorist bears, but it is equally one which confronts the two-factor theorist, since she argues that there is some cognitive feature of subjects with delusions which is *abnormal* and differentiates them from the non-delusional population. It thus equally falls on her to demarcate the scope of the normal range, since it is she who posits *non-standard* belief formation or evaluation processes. Here is one way of thinking about the issue: take the cognitive contribution operating in subjects with delusions, and consider whether it involves mental processes that are significantly different from those exhibited in the general population. Now recall that the cognitive contribution operates in a population of subjects who undergo anomalous experiences. Now ask: are we required to characterize the causally contributory psychological influences on delusion formation as *non-standard*? Especially once we reflect on the context of anomalous and distressing experience to which these causally relevant psychological influences are applied? My view is that there is no reason to think that we should. It is entirely consistent with a one-factor theory that there are subjects who have anomalous experiences who nevertheless form different beliefs. When we fail to keep normal range individual psychologies fixed, we may well find cases of subjects with delusions with the same experience as subjects without delusions.

Note that a one-factor theorist does not deny that there can be cognitive influences such as background beliefs and proneness to certain thinking styles. However, theories of delusion formation aim to pick out what is *different* between subjects with delusions and subjects without, *which is explanatorily relevant to delusional belief formation*

*as opposed to non-delusional belief formation.* It is for this reason that the second factor must be one that is unique to folk with delusions (as recognized by two-factor theorists, see Davies et al. 2005: 228 who take the second factor to be ‘a departure from what is normally the case’; McKay et al. 2010: 316–17 who take the deficit two-factor approach as one which ‘conceptualises delusions as involving dysfunction or disruption in ordinary cognitive processes’; and Stone and Young 1997: 342, in their talk of delusional reasoning being ‘abnormal’, and ‘differences between people with and without delusions’). The objective in this debate is not to give a full causal map of *everything* involved in delusion formation. If it were then *of course* we would need to focus on more than one ‘factor’, we would also need to talk about the subject having been born, there being oxygen in her environment, her being able to form beliefs, and so on. There would likely be thousands of ‘factors’ involved in such an account, depending on the preferred fineness of grain. That these ‘factors’ are causally involved in delusion formation is not where the dispute lies, since what we mean by *factors* is not merely something which plays some causal role in delusion formation. Rather, the term picks out that contribution having the feature of being abnormal and hence explanatorily relevant to the distinction between delusional and non-delusional beliefs.

What if a two-factor theorist were to grant that the cognitive contribution she identifies as involved in delusion formation is statistically abnormal but is also found among the non-delusional population? It is not my view that this contribution would be of no interest to those interested in the kinds of cognitive features which tend to be involved in delusion formation. But this is a development of a one-factor approach, not an alternative to it. The one-factor view is perfectly compatible with there being non-ideally rational cognitive contributions occurring in delusion formation, that indeed, are partly explanatory of delusion formation. But if such contributions are not distinctive of *delusional* belief formation, they are perfectly acceptable to a one-factor framework.

What one- and two-factor theorists disagree on is whether the background psychologies which help prompt or maintain the delusional explanation for a strange experience are constitutive of a second abnormality. The two-factor theorist posits a nonstandard psychology with respect to belief formation or evaluation present in subjects with delusions. To posit a second factor is to make a substantive point, and, I say, a substantive error.

### **3. Alien abduction belief**

I will refer to subjects who believe they have been abducted by aliens as *abductees* (where that term is not factive). In what follows I describe the kinds of experiences abductees have and the kind of narrative they typically report.

Psychologists looking for a naturalistic explanation of these beliefs have appealed to awareness during sleep paralysis (ASP) and accompanying hallucinations. During Rapid Eye Movement (REM) sleep, the sleeper is immobilized, that is, motoric output is blocked. In ASP, the sleeper awakes before the paralysis has disappeared and becomes aware that they are unable to move (McNally and Clancy 2005: 114). ASP can be accompanied by an increase in heart rate, difficulty breathing, and feelings of dread (Holden and French 2002: 166).

ASP is relatively common in the general population.<sup>13</sup> Richard McNally and Susan Clancy put the figure of those who have experienced one or more episodes of ASP at approximately 30% of the general population. ASP can be accompanied by hypnogogic (sleep onset) and hypnopompic (sleep offset) hallucinations. Of those subjects who experience ASP, 75% of them will hallucinate in at least one modality during the ASP experience (2005: 114). Though this only lasts a few seconds or minutes, it can be a very frightening experience (McNally and Clancy 2005: 114, see also Cheyne et

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<sup>13</sup> Cheyne and Pennycook note 'substantial variance' on the prevalence of sleep paralysis (estimates between 5% and 60%). They suggest this variance is likely reflective of 'differences in survey methods, operational definitions, age and other demographic characteristics of survey participants, and cultural factors' (2013: 135).

al 1999; Sharpless et al 2010). Hallucinations which accompany ASP have been described as falling into one of three categories: *intruder* (experience of a presence in the room), *incubus* (pressure on the chest and difficulty breathing), and *vestibular-motor* (feeling of flying, floating, spinning, or being out of one's body) (Cheyne and Pennycook 2013: 135–6). Abductees report a variety of these experiences; the hallucinations may be visual, including 'lights, animals, strange figures, and demons', or auditory including 'heavy footsteps, humming or buzzing noises' (Holden and French 2002: 167). Consider the following account of such an episode:

Imagine opening your eyes shortly before dawn, attempting to roll over in your bed, and suddenly realizing that you are entirely paralyzed. While lying helplessly on your back and unable to cry out for help, you become aware of sinister figures lurking in your bedroom. As they move closer to your bed, your heart begins to pound violently and you feel as if you are suffocating. You hear buzzing sounds, and feel electrical sensations shooting throughout your body. Within moments, the visions vanish and you can move again. Terrified, you wonder what has just happened. (McNally and Clancy 2005: 114).

We see these kinds of experiences in two representative reports from McNally and Clancy's abductees (2005: 116):

A female abductee was lying on her back when she woke up from a sound sleep. Her body was completely paralyzed and she experienced the sensation of levitating above her bed. Her heart was pounding, her breathing was shallow, she felt tense all over. She was terrified. She was able to open her eyes, and when she did so, she saw three beings standing at the foot of her bed in the glowing light.

A male abductee awoke in the middle of the night seized with panic. He was entirely paralyzed, and felt electricity shooting throughout his body. He felt his energy draining away from him. He could see several alien beings standing around his bed.

An experience of the above kind can lead the subject to seek an explanation, especially since, though the combination of ASP and hallucination is relatively common, knowledge of these states is not, making it unlikely that the experience would be explained by appeal to them (Holden and French 2002: 166).<sup>14</sup> Some may shrug off the experience as inexplicable, others come to believe that they were abducted by aliens (or whatever fits the cultural narrative, see Hufford 1982: 2005).

McNally describes the progression of a typical case as follows: a subject wonders what happened, begins to read about these experiences, sees therapists who endorse the abduction explanation, and enters into memory recovery sessions in which under hypnosis they may generate more details about the experience and what happened thereafter. This can include being 'whisked through walls up into the sky into spaceships', being 'sexually probed by aliens', and 'involved in hybrid breeding experimentation' before being 'brought back down to the bedroom, before the break of dawn' (McNally 2014).

Abduction beliefs formed on the basis of these experiences demand attention from theorists of delusion formation. Once we attend, lessons can be learned. Before giving an explanation of abduction beliefs which does not appeal to cognitive abnormality, I first turn to the question of whether these beliefs are delusions.

#### **4. Are abduction beliefs delusions?**

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<sup>14</sup> Kevin Sharpless and Jacques Barber (2011) argue that ASP ought to be more regularly assessed, since several studies have found that subjects experience clinical relief when their experiences are normalized (see Otto et al. 2006; Sharpless et al. 2010).

Abduction beliefs vary in respects relevant to whether they are properly characterized as delusions, the category is heterogeneous and so resists a general classification. Let us start by asking whether abductees *believe* their claims, since if they did not then what we learn from this case could not tell us much about *delusional belief* formation. Here are two reasons to think that they do. First, abductees present as sincerely avowing their abduction attitudes (French 2001; cf. Baker 1992). Given that we can explain their experiences by appeal to ASP and hallucination (indicating the possibility of such experiences), we are, all else equal, justified in taking the experience reports as genuine. Second, research into the physiological responses of abductees when listening to recordings of themselves reporting their abduction experience has shown that their responses were 'larger than the responses of PTSD patients to the scripts of their traumatic experiences' (McNally and Clancy 2005: 117). These findings are taken to 'underscore the power of emotional belief', and the psychophysiologic profile of abductees will 'resemble that of PTSD patients even if they themselves do not qualify for the diagnosis' (McNally and Clancy 2005: 117).<sup>15</sup> We can be a little more careful here and say this: if part of what explains the reactions of PTSD subjects to scripts of their traumatic experiences is that they believe that that experience really occurred, then if abductees' reactions to hearing scripts of their experiences are comparable, this suggests that abductees believe that their experience really occurred.

Though it is difficult to pin down what makes something a *delusional* belief, as we saw earlier, we can point to the epistemic surface features delusions possess, to get some grip on what characterizes these states. Characterizing delusions in such terms stacks the deck against accounts such as mine which resist the claim that delusions are the result of abnormal reasoning from experience to belief. Nevertheless, features such as *resistance to counterevidence* may well be characteristic of delusions, just so long as we do not take

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<sup>15</sup> Even if abductees did warrant a diagnosis of PTSD, this would not be to introduce a second factor, since any clinically significant feature of their cognition would not be along the appropriate dimension (i.e. with respect to belief formation or evaluation).

such resistance to be qualitatively different from the resistance to having one's beliefs challenged that occurs within the normal population. With that caveat, the epistemic features of delusions are had by at least some abduction beliefs, and so we have reason to think that some of them are delusions. Clancy notes that:

Once the seed of belief was planted, once alien abduction was even suspected, the abductees began to search for confirmatory evidence. And once the search had begun, the evidence almost always showed up. [...] Once we've adopted the initial premises ("I think I've been abducted by aliens"), we find it very difficult to disabuse ourselves of them, they become resilient, immune to external argument. (Clancy 2005: 51)

Abduction beliefs are also often accompanied by distress and preoccupation (Clancy 2005: 138-9), as we learned from McNally and Clancy's studies on abductees and their physiological profiles. This is a feature of belief which 'increases the probability that it will be described as delusional' (Garety and Freeman 1999: 114). Finally, the evidence abductees have does not properly support the content of their beliefs, since often there are alternative (and better) explanations available to them. Abductees are often aware of naturalistic explanations of their experiences, but do not adopt these explanations.

So far we have seen reasons for thinking that abduction attitudes are beliefs, and that in some cases at least, they are delusional beliefs. However, it is worth addressing the claim that they are not in fact delusions. If they are not, the two-factor theorist may claim a license to properly cast them as outside of her remit. The claim that abduction beliefs are not delusions requires a principled reason why not which does not beg the question against my account in favour of the two-factor theory. Specifically, the reason cannot be that these beliefs are not delusions because they are not formed via abnormal belief formation processes.



Suppose that such a reason can be given, and abduction beliefs can be carved out of the domain of delusions. It might now be wondered what makes these beliefs such a good case to illuminate the debate between one- and two-factor theorists. I focus on abduction beliefs (over, say, prejudiced beliefs, religious beliefs, etc.) for three reasons. First, abductees have strange experiences, a factor all empiricists agree on. Second, at least some two-factor theorists think abduction beliefs are delusions, and so already take themselves to bear the burden of accounting for them (Coltheart 2005b). Third, as noted earlier, theorists interested in explaining abduction beliefs recognize that abduction experiences are not unique to abductees, but they do not seek to identify a clinically significant cognitive contribution to do what remains of the explanatory lifting. It might then be asked whether this methodology would be appropriate to explain monothematic delusion formation. I argue that it is. Of course, sometimes prejudiced and religious beliefs *are* delusions when they start to have the features outlined earlier, and such cases would also be illuminating. Abduction beliefs have such features more often and so lessons can be learned from this case.

Abduction beliefs are relevantly similar enough to delusions such that our story of their etiology can inform our account of monothematic delusions proper.<sup>16</sup> The case can shed light on the delusion formation debate by demonstrating that a rather bizarre belief can be generated (or not) through the combination of experience and normal range cognitive influences. Once we hold individual psychology fixed (that is, cognitive variations within the normal range), the explanatory work gets done by the experience. If abduction beliefs are not *delusions*, this point remains. If they are, the one-factor account

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<sup>16</sup> Just as it need not be the case that abduction beliefs are properly characterized as delusions to be informative, nor need it be the case that they are properly characterized as *monothematic* delusions. However, for completeness, there are good reasons for thinking that they can be more narrowly described in this way. Monothematic delusions, as noted earlier, have highly specific contents and present against an otherwise unremarkable set of background beliefs (*unremarkable* need not be *rational*). Second, Coltheart takes these beliefs to be within his two-factor remit which is an account of monothematic delusion formation.

can explain them, and this explanation can generalize to other delusions (as I will argue, §7).

To sum up: if abduction beliefs are not delusions, they nevertheless demonstrate that there can be strange experiences and other influences combined in different ways resulting in a bizarre belief (or not), but that it is the experiential component which does the explanatory work, when we hold normal range individual psychology fixed. Below I argue that the ways in which abduction beliefs are different from monothematic delusions proper does not make a difference to the best account of their etiology. If abduction beliefs are delusions, then a one-factor account can explain their formation, and this framework can generalize to other monothematic delusions. From here on I will speak of abduction beliefs as delusions for ease of exposition.

## **5. Explaining abduction belief**

In this section I argue that the only abnormal thing we need to appeal to is the experiences abductees have in order to explain their belief. A one-factor framework can give the following explanation: the abductee has a strange experience which she cannot explain, creating in her ‘puzzlement, anxiety, and a search for an explanation’ (Maher 2006: 181). In particular, she comes to believe something which explains her experience, and this belief serves ‘the purpose of providing order and meaning for empirical data obtained by observation’ (Maher 1988: 20). The way in which she forms this belief differs in no respect from the ways in which the beliefs of normal subjects are formed. I take it, also, that the maintenance of abduction beliefs are explained in just the same way as the maintenance of other beliefs, and unless and until there is evidence from the study of abductees to suggest otherwise, that is how we should approach understanding their formation.

The question now is whether this sketch of an explanation is adequate, or whether we need also appeal to something cognitively amiss in abductees (as two-factor

theorists claim necessary to explain monothematic delusions). Earlier we saw some versions of the two-factor account, according to which the second factor is a data-gathering bias (Garety and Freeman 1999), the privileging of observational data (Stone and Young 1997), or an inability to inhibit a pre-potent doxastic response (Davies et al. 2001). From here on I focus my discussion on Max Coltheart's two-factor view, for two reasons. Firstly, Coltheart has discussed abduction belief, taking it as a burden on the two-factor account. Secondly, Coltheart argues that the second factor is one caused by neurological damage, and this has muddied the waters in the debate, since a neurological abnormality has been taken to straightforwardly establish an abnormality of the relevant kind. It is very important to note that what I say here with respect to the putative second factor is applicable to other two-factor accounts, insofar as the outcome of the discussion is that the one-factor account has the resources to explain abduction beliefs, and so a two-factor account is not required. This is the key point of the paper.

As noted earlier, Coltheart's second factor is 'a (very poorly specified) defect of a belief evaluation system', located in the right hemisphere (Coltheart 2005a: 154).<sup>17</sup> Coltheart notes that this second factor (which he takes to be causally implicated in all monothematic delusions), is absent in abductees (Coltheart 2005b: 75, see also Coltheart, Langdon, and McKay 2011: 291), and so we need to identify an alternative second factor. His discussion is similar to the objection against the one-factor account discussed earlier, namely, we need to identify a second factor which reveals the difference between someone who has an anomalous experience and does not become delusional, and someone who has that same kind of experience and does. I argued that the two-factor theorist makes a mistake here in expecting the one-factor theorist to identify a particular kind of anomaly which would, *whatever the psychology*, give rise to delusion.

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<sup>17</sup> Neuropsychological support from this can be found, for example, in Burgess and colleagues' (1996) literature survey of twenty-two cases of delusion in which brain damage was detected, for discussion see Coltheart (2007: 1051-2).

Not everyone who experiences sleep paralysis seeks an explanation, some subjects 'shrug it off as a weird and inexplicable occurrence' (McNally and Clancy 2005: 120). The two-factor theorist may demand an explanation of why some people are able to shrug the experience off, and others opt for the explanation that they have been abducted by aliens. In this vein, Coltheart cites Clancy and colleagues' study of eleven abductees, with all eleven reporting that 'they had begun to suspect that they had been abducted after a sleep episode characterized by awakening, full body paralysis, intense fear, and a feeling of presence', with several participants reporting 'tactile or visual sensations (i.e. levitating, being touched, seeing shadowy figures)' (Clancy et al. 2002: 456). Coltheart notes that this kind of experience is not uncommon, citing J. Allan Cheyne and colleagues' survey of 870 university students. 22.5% had experienced sleep paralysis coupled with hallucinations. Coltheart notes that he is 'going to assume' that the percentage of people with abduction delusion 'is vastly lower than 22.5%' (2005b: 75). If this is right, we have a version of the objection to the one-factor account as applied to abduction belief since 'there will be many people who have experienced sleep paralysis with hypnogogic or hypnopompic hallucinations yet do not have the alien abduction belief' (Coltheart 2005b: 75).

A one-factor account can explain the difference in resulting beliefs upon the same experience by appeal to individual differences in background psychologies which fall within the normal range. Recall that the view that mechanisms of belief formation and evaluation of individuals with delusions operate within the normal range is consistent with various outcomes with respect to beliefs formed upon certain experiences.<sup>18</sup>

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<sup>18</sup> An alternative response is available. If one thought the anomalous experiences of abductees were *sufficient* for abduction belief, one could argue that the purported same experience in the absence of abduction belief is in fact not the same experience. Maher responds thus to an equivalent objection, and this line can be taken in response to the general problem (see fn. 11). This response might be especially plausible in the case of abduction belief, since often the content of the belief (especially details about medical procedures, reproductive practices, and being aboard a spaceship), comes about after memory recovery sessions. If one shrugs off a nighttime experience, one is unlikely to end up in memory recovery sessions. If part of the experience is the recovered memories, one does not have the full experience required for abduction belief. Another version of this response is to claim that the

## 6. Going Disjunctive

I now note an additional issue presented by the case of abduction beliefs. If they are delusions, the two-factor theorist must find a new second factor to accommodate this particular case, since previously identified second factors are simply not to be found in abductees. The case of alien abduction belief thus forces two-factor theorists to go disjunctive about the second factor.

Staying with Coltheart's view, we have no reason to believe that the neural basis for the proposed second factor is present in abductees (Coltheart 2005b: 75). In addition, research on abductees has shown little psychopathology, Holden and French found 'no convincing evidence for higher rates of serious psychopathology amongst abductees compared to the general population' (2002: 163). Indeed, abductees are little different from non-abductees along several other dimensions, including emotional intelligence, openness, agreeableness, extraversion, and fantasy proneness<sup>19</sup> (Hough and Rogers 2007: 154–7).

To this two-factor theorists might say that abduction beliefs are not delusions, and it is for this reason that two-factor theories cannot offer the right story. However, Coltheart for one takes abduction beliefs to be within his remit, and we have seen some reasons for thinking these beliefs are delusions (§4), and so an explanation of these cases must be given. It has been recognized that 'the two-factor account of delusional belief is not intrinsically committed to the idea that these factors stem from neurological damage'

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experiences of the subjects who form abduction beliefs are different from the experiences of those who do not, with respect to the intensity of fear and feeling of threat felt during the experience (that is, it is higher in those who become abductees). Evidence for such differences comes from work on the reported fear and feelings of threat during the sleep paralysis experience, and reporting of supernatural beliefs regarding the cause of the experience (Cheyne and Pennycook 2013: 142).

The success of these responses depends on determining whether there are subjects who have had the same kind of anomalous experience (complete with relevant memories), who nevertheless do not believe that they were abducted. Such subjects would demonstrate the inadequacy of this response. My preferred response is not hostage to empirical fortune in this way.

<sup>19</sup> Though there is as yet no consensus on whether abductees exhibit a higher level of fantasy proneness. Several researchers in addition to Hough and Rogers above have suggested not (Rodehier et al. 1991; Ring and Rosing 1990; Patry and Pelletier 2001), though others have suggested so (French et al. 2008; Bartholomew et al. 1991).

(Coltheart, Langdon, and McKay 2011: 291). However, although the second factor need not stem from damage, two-factor theorists have claimed that a *particular* second factor is common to all delusions. In Coltheart's case, it is the 'imperviousness to counter-evidence' which prevents the hypothesis generated by the anomalous experience from being rejected (Coltheart 2010: 18).

In the case of abductees however, Coltheart's proposed second factor is not understood in these terms. Instead, Coltheart points to work showing that abductees show higher levels than the general population on some measures. For example, McNally and Clancy found that abductees scored higher on the Dissociative Experience Scale (DES), the Absorption Scale, and the Magical Ideation Scale than controls, where the

DES taps alterations in consciousness ranging from the ordinary (e.g. 'spacing out' on a long car trip) to the bizarre (e.g. not recognizing one's reflection in the mirror). The Absorption Scale taps the ability to become engaged in imaginative experiences (e.g. reading a novel) – a trait linked to fantasy proneness. The Magical Ideation Scale assesses belief in unconventional phenomena (e.g. telekinesis, reincarnation). (2005: 116–17)

McNally and Clancy's abductees also entertained 'a wide range of "New Age" beliefs (e.g. astral projection, foretelling the future) that might have made them especially prone to endorse an alien encounter interpretation of their sleep paralysis episodes' (2005: 120).<sup>20</sup>

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<sup>20</sup> An alternative route to explaining the adoption of abduction beliefs is by appeal to individual differences in memory. There is some research which has shown that certain subjects (including abductees) are more prone to the Deese/Roediger-McDermott (DRM) associative memory illusion, where one falsely remembers stimuli that were not present in a list (for an overview see Gallo 2010). There is evidence that some of the processes which contribute to this illusion are also involved in false autobiographical memories (Clancy et al. 2002). Note that these differences are characterized as

More generally, subjects with paranormal beliefs do less well at syllogistic reasoning, see meaning where there is none due to a distorted concept of randomness, and are more susceptible to anomalous experiences. There is also evidence, though mixed, that subjects who have paranormal beliefs perform poorly in critical thinking tests (French and Wilson 2007: 3), and that they are less successful on tasks of probability estimation than non-believers (Blackmore and Troscianko 1985).

What Coltheart says about the generation of abduction beliefs is the kind of thing we should say about monothematic delusions more generally: abnormal experience plus some normal range irrationality contribute to the formation of a bizarre belief.<sup>21</sup> With respect to this kind of framework, Coltheart has suggested that we ought not count New Age believers as part of the ‘healthy population’:

New Age people who believe in UFOs and so on—that doesn’t sound very healthy to me. So they’re healthy in the sense that they don’t have brain damage but they’ve got a very strange belief system, it’s very unlike most people’s belief system, and if they didn’t have that belief system, then they wouldn’t become delusional. (Coltheart 2014)

There is a dubious statistical claim here—that an abductee’s belief system is unlike ‘most’ other people’s. However, even granting that does not get Coltheart to a second factor. Being atypical is not the same as being found in all and only the deluded. The kinds of cognitive contributions to belief formation Coltheart points to in order to explain abduction beliefs are present in the ordinary population, and are just the kind of thing

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‘individual differences’, and not in terms of pathology. So it is consistent with a one-factor explanation of abduction beliefs that there are these processes, since they are normal range ones.

<sup>21</sup> See Coltheart, Langdon, and McKay (2011: 291), who suggest a predisposition to accept New Age beliefs is the second factor, but is also one which is ‘present to varying degrees in the general population’. As I noted earlier, this characterization of further cognitive contributions to delusion formation makes the view not a competitor to the one-factor account, but a development of it. To be constitutive of a second factor, this predisposition must be an abnormal one, as Coltheart suggests in the quotation below.

which represent differences along the normal range. We have seen that abductees may score higher than many on absorption, dissociation, and magical ideation, but these are cognitive features shared by subjects who are not abductees. We can get a full explanation of why abductees believe as they do by appeal to their strange experiences, and individual differences in reasoning styles which prompt and maintain explanations of experiences in these terms.

The case of abduction belief underlines my point that if we fail to keep individual psychology fixed, then we will find that there are cases of subjects with delusions with purportedly the same experience as subjects without delusions. We ought not take variations in normal range human psychology which facilitate one becoming delusional upon certain experiences as indicative or constitutive of a second factor. The case of abduction belief brings this out: here we have a case where there is no cognitive abnormality, but there is an anomalous experience and individual differences along the normal range which contribute to the formation of a bizarre belief. We ought not factorize normal (even if not ideally rational) cognitive processes.

The case of abduction belief then has highlighted the importance of normal range (if irrational) contributions to bizarre belief, and also forced a disjunctive two-factor theory to accommodate the case. Coltheart's two-factor account of abduction belief is that the thinking style found more often in abductees should not be described as a second *deficit*, 'but it might be thought of as a second factor that, when combined with the first factor [...] creates the alien abduction delusion' (Coltheart 2005b: 76). Coltheart is here forced to a disjunctive account of the second factor: it is a neuropsychological deficit which impairs belief evaluation *or* it is a range of thinking styles.<sup>22</sup> Furthermore, there is a large range of cognitive contributions, and so in addition to the theoretical cost of going

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<sup>22</sup> I have been working with statistical abnormality in this paper (see fn. 9). However, there may also be a slide from functional abnormality to statistical abnormality here. Coltheart wants to deny that the causal contributions to abduction belief are *deficits*, but not deny that they are *factors*. So Coltheart has to go disjunctive in identifying a different causal contribution, but also in capturing the nature of the contribution in its departure from normality.



disjunctive, it might be very difficult indeed to identify a single one of these contributions as *the* second factor.

In contrast, the one-factor theorist is not forced to recognize a difference between abduction beliefs and other monothematic delusions. The two-factor theorist thus runs into trouble with Ockham's razor in two ways: First, all two-factor theorists say there is a second factor. Second, unless and until there is evidence that abductees exemplify belief formation or evaluation in line with their putative second factor, two-factor theorists commit a second infraction in the case of abduction belief by proposing that there is a *different* second factor. Not only do two-factor theorists multiply factors beyond necessity, they sometimes multiply the number of unnecessary second factors.

## 7. Generalizing the point

If we allow a one-factor account of abduction belief, we ought also allow a one-factor account of (other) monothematic delusions. This is not to make the weaker claim that if a one-factor account is appropriate in the former case, then we can see whether it works in other cases. It is not merely that a generalized one-factor account ought not be *ruled out*. Rather, my claim is that *if* a one-factor account works for abduction belief, we have a case from which to generalize to an account of monothematic delusions simpliciter. This conditional is true since there are no differences between abduction beliefs and (other) monothematic delusions which block the one-factor account in cases of the latter, and so we have not yet been given a reason for the two-factor account to be *ruled in*. In this section I consider two differences which put pressure on the proposed generalization and argue that they are not relevant ones.

The first difference is one I flagged earlier: many subjects with monothematic delusions have damage to the right lateral pre-frontal cortex which is hypothesized to be involved in belief evaluation (Coltheart 2005a: 154). This is absent from abductees. This fact may be thought to block the generalization of the one-factor account of abduction

belief to a one-factor account of monothematic delusion simpliciter. Abduction beliefs might then be considered a distinct case, importantly different from other monothematic delusions, and any theory of their formation simply cannot be generalized.

The presence of this neurological damage, and its proposed role in delusion formation and maintenance raises three challenges to my one-factor theory as applied to monothematic delusions. First, its presence might be thought to straightforwardly establish a second abnormality, falsifying the one-factor theory. Second, the two-factor theorist can grant the role of the reasoning style introduced by this damage, the one-factor theorist cannot. Third, we might ask how the one-factor theorist can explain the co-existence of brain damage and monothematic delusions? How can she explain why there is this common locus of brain injury across a variety of otherwise disparate monothematic delusions? I will respond to these challenges in turn.

To the first challenge: though there is a second abnormality here, this is not inconsistent with the one-factor theory, since establishing a *neurological* abnormality is not to establish an abnormality at the right level of explanation. People may vary in the normal range with respect to reasoning for a number of reasons, the explanation of some instances of variation may go via appeal to this neurological damage. It might well be the case that certain kinds of damage make people more susceptible to delusions than others, but it is only if the damage introduces belief formation or evaluation *outside of the normal range* that it is problematic for the one-factor theory. So the observation of neurological damage does not show that an abnormal form of belief formation or evaluation needs to be posited.

The second challenge was that the one-factor theorist is unable to account or allow for the role of this neurological damage. However, I do not claim that this damage plays no causal role in the occurrence of delusions. Rather, my claim is that even if it affects reasoning, it remains to be shown that it does so in a way that represents a departure from the normal range. Thus it is left open both that delusions may occur in

someone without the damage, or someone with such damage may not go on to develop the delusion (perhaps in the absence of an anomalous experience). Neurological damage simply does not speak to there being a second factor in the sense relevant to the debate. The burden is on the two-factor theorist to make good on the claim that neurological damage entails abnormal reasoning. This is quite an ask, and involves, at the very least, properly distinguishing normal from abnormal reasoning.

The third challenge was to *explain* the presence of this damage. However, this is not the burden of either one- or two-factor theorists. The task is to explain the formation and maintenance of monothematic delusions, it is not to explain the presence of neurological damage which is sometimes (or even usually) associated with such delusions. The only burden on theories of delusion formation and maintenance is to be *consistent* with these findings, and to accord them the explanatory power they are due. I do not deny the contributory role this damage might play, I only deny that it has been shown to have the feature of being clinically abnormal in the relevant sense.<sup>23</sup>

I underline the point that my view is not that it is *normal* to have right lateral pre-frontal cortex damage. Brain damage is not itself normal but it might not be so severe as to put a subject outside of the normal range on the relevant *psychological dimension*. Indeed, my view is even consistent with the claim that the damage puts a subject outside of the normal range on *some* psychological dimensions, but it has not been shown that it does so with respect to this particular one (belief formation/evaluation). Normalizing

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<sup>23</sup> An analogy speaks to challenges two and three. Imagine a two-factor theory which identified the second factor as the presence of oxygen in a subject's environment. Now imagine I argued along the following lines: 'oxygen in the environment is of course relevant to a full explanation of delusional belief formation, but its contribution does not represent a departure from normal range belief formation with respect to the role played by oxygen inhalation'. An objector responds: 'how do you explain the presence of oxygen in the environment in all cases of monothematic delusion? And how do you explain the role of oxygen in the way the two-factor account can?' She who denies that oxygen is a *factor* does not have to say that it plays no role, only that it does not play a role which represents departure from the normal range along this dimension. Nor is she burdened with explaining its presence (though her theory must be consistent with evidence of its presence). With respect to challenges two and three, my position is no more troubled by the presence of neurological damage to areas of the brain involved in belief evaluation than it is by the presence and contributory role of oxygen in delusion formation.

neurological damage is no part of my view, the point is rather that such an abnormality does not entail abnormality at the level of belief formation or evaluation.

How does the one-factor theorist explain what is going on with subjects who have an anomalous experience associated with a certain delusion, but do not become delusional? If it is not a second factor which explains the difference what does? To this I say: a variety of things, and the cognitive processes which contribute to the formation of the delusion are not qualitatively different from the cognitive processes which go on in belief formation in the non-delusional population. Subjects with delusions might reason in particular ways because of pre-frontal cortex damage, but that is compatible with that reasoning being inside of the normal range (even if the cause of the reasoning is not normal). Neither the absence of neurological damage in abduction belief, nor the presence of it in (other) monothematic delusions, require us to identify a second factor causally implicated in the formation of delusion in the second case.

I turn now to the second difference between abduction belief and other monothematic delusions which the two-factor theorist might argue blocks the generalization of the one-factor theory from the former to the latter. Abduction belief might be thought to require a New Age background theory. If that is right, then those who end up with abduction beliefs begin with a high subjective probability in the possibility of abduction, making the best explanation of their experiences one in terms of abduction. Similar things cannot be said about, for example, Capgras delusion. Those who experience a lack of affective response to a loved one, do not have high subjective probabilities in the possibility of imposters. That means the belief formation story between these two cases must be very different—one is predicated on a background theory, the other is not.

I do not deny that there might be this difference (it has not been shown that a New Age background theory is *required* for abduction belief, but suppose it is). However, that does not speak against the generalization from an account of abduction belief to an

account of other monothematic delusions. The claim is not that abduction beliefs are formed *in the same way* as other monothematic delusions, the claim is only that the cognitive contributions present in this case can take one from a strange experience to a bizarre belief, and that that structure may well be present in the case of delusions. There are all sorts of ways one can vary within the normal range of cognitive contributions, and it is the move from strange experience to bizarre belief with the help of these normal range cognitive contributions which is being generalized to monothematic delusions. It is no part of my view that there is a *particular* cognitive contribution present across cases, my claim is only that we need not suppose that *whichever* cognitive contribution there is, it is an abnormal one.

## 8. Conclusion

I argued that the formation and maintenance of abduction beliefs can be explained by a one-factor account, that is, one which foregoes any appeal to cognitive abnormality at the level of belief formation or evaluation. The explanatory virtues displayed by the one-factor account are not limited to this case, but generalize, since there are no known differences between abduction beliefs and monothematic delusions which indicate the need for additional explanatory factors in cases of the latter. Though monothematic delusions are associated with damage to the right lateral pre-frontal cortex, this does not speak against the one-factor account. Nor does its absence in abductees block generalizing a one-factor view from these cases to monothematic delusions simpliciter. In addition, the case of alien abduction belief forces the two-factor theorist to go disjunctive about the second factor to accommodate this case.

The research orientation of theorists interested in abduction beliefs has been to identify which normal range processes may be involved in their formation. This research methodology is one which theorists interested in monothematic delusions should consider, and we ought to understand the putative second factors identified by two-

factor theorists as ways in which subjects might vary within the normal range. I do not rule out that there could be some cases where a second factor is implicated, but such cases (if there are any) should not be taken to tell us anything about delusional belief formation more generally. Rather, the one-factor position ought to be the default approach to understanding delusion formation. I conclude that researchers interested in delusion have much to learn from the case of abduction belief, specifically, that it supports a one-factor account of monothematic delusion formation. To put it in the crudest terms, in the presence of anomalous experiences, it is normal for humans to have bizarre beliefs.

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